JACC Vol. 22, No. 2
August 1993:361–7

Long-Term Prognostic Significance of ST Segment Depression During Acute Myocardial Infarction

RONALD J. KRONE, MD, FACC, HENRY GREENBERG, MD, FACC,*
EDWARD M. DWYER, JR., MD, FACC,* ROBERT E. KLEIGER, MD, FACC,
WILLIAM E. BODEN, MD, FACC† AND THE MULTICENTER DILTIAZEM POSTINFARCTION TRIAL RESEARCH GROUP

St. Louis, Missouri; New York, New York; Boston, Massachusetts

Objectives. **The purpose of this study was to evaluate the long-term prognostic value of ST segment depression on the electrocardiogram (ECG) in patients with acute myocardial infarction.**

Background. **The prognostic importance of ST segment depression on the ECG has been studied in small groups of patients with infarction, but larger numbers are needed.**

Methods. **Coronary care unit ECGs of 1,234 patients who survived the coronary care unit with acute Q wave (n = 896) or non-Q wave (n = 338) myocardial infarction were analyzed for the presence of ST segment depression. Patients were followed up for up to 4 years.**

Results. **ST segment depression was present in 607 patients. Those with ST segment depression had a 1-year mortality rate of 10.3% compared with a rate of 5.6% for those without ST segment depression (p = 0.002). This effect was seen in both the Q wave and non-Q wave subgroups. Of the 437 patients with anterior ST segment elevation, those with ST segment depression in other regions had a 13.6% 1-year mortality rate compared with a rate of 6.9% for those with no ST segment depression (p = 0.0005). Of the 514 patients with inferior ST segment elevation, those with ST segment depression in other leads had an 11.0% 1-year mortality rate compared with a 1.8% rate for those with no ST segment depression (p = 0.0001). The Cox proportional hazards model showed that ST segment depression was an independent predictor of mortality over the follow-up period.**

Conclusions. **ST segment depression on the admitting ECG in patients with acute myocardial infarction is a predictor of increased mortality in the year after infarction.**

(J Am Coll Cardiol 1993;22:361–7)

Stratification of patients with acute myocardial infarction into high and low risk subsets is essential for sound clinical decision making. This is especially important early, when decisions concerning thrombolytic therapy and acute mechanical revascularization must be made (1).

The presence of ST segment depression alone or in combination with other electrocardiographic (ECG) findings has been shown to indicate a higher long-term mortality rate after infarction (2–7); however, although the value of reperfusion therapy in patients with acute infarction demonstrating ST segment elevation on the ECG has been clearly established, its efficacy in patients with ST segment depression remains ill defined (8–11). The reasons for this apparent paradox are not clear. The Multicenter Diltiazem Postinfarction Trial (MDPIT) enrolled 2,466 patients soon after acute myocardial infarction, and a detailed analysis of the qualifying ECG was performed. The present report utilizes the extensive MDPIT data base to examine the value of ST segment depression on the coronary care unit ECG as a predictor of cardiac events and mortality after hospital discharge to begin to understand these apparent divergent responses to treatment.

Methods

Study patients. The MDPIT was designed to evaluate the protective effect of prophylactic diltiazem on cardiac events after acute myocardial infarction. The details of the organization of the trial, patient recruitment, follow-up, end point analysis, data management and statistical methods have been published elsewhere (12). Patients at 38 hospitals at 23 centers in North America were enrolled between 1983 and 1986. The follow-up period ranged from 12 to 62 months (average 25). Patients from 25 to 75 years of age, who were discharged alive from a coronary care unit after enzymatic confirmation of acute myocardial infarction, were eligible for

From the Washington University School of Medicine and the Jewish Hospital of Saint Louis, Saint Louis, Missouri; *Saint Luke's–Roosevelt Hospital Center and Columbia College of Physicians and Surgeons, New York, New York; †Boston Veterans Affairs Medical Center, Boston, Massachusetts. This study was supported in part by a consortium grant from Godecke Aktiengesellschaft (Freiburg, West Germany), Laboratories de Dr. Esteve, SA (Barcelona, Spain), Marion Laboratories, Inc. (Kansas City, Missouri), Nordic Laboratories, Inc. (Laval, Quebec, Canada), Lars Synthelabo (Paris, France), Tanabe Seiyaku Co., Ltd. (Osaka, Japan) and Warner-Lambert International (White Plains, New Jersey).

Manuscript received June 29, 1992; revised manuscript received January 19, 1993, accepted January 22, 1993.

Address for correspondence: Ronald J. Krone, MD, Department of Cardiology, The Jewish Hospital at the Washington University Medical Center, 216 S. Kingshighway, Saint Louis, Missouri 63110.

0735-1097/93/$6.00

enrollment. Acute ECG changes were not required for admission.

Patients were excluded for any of the following reasons: cardiogenic shock or symptomatic hypotension, pulmonary hypertension with right ventricular failure, second- or third-degree heart block, rest bradycardia, inability to take diltiazem, Wolff-Parkinson-White syndrome, high likelihood of requiring cardiac surgery or presence of any noncardiac condition associated with decreased likelihood for survival. Of particular importance for this study was exclusion of patients requiring or likely to require treatment with calcium channel blocking agents for conditions such as severe angina uncontrolled by other medications. The study group consisted of the 1,234 patients randomized to placebo medication.

Data acquisition. Coronary care unit ECGs were coded according to the previously published Manhattan classification (13). Two ECGs, one obtained in the coronary care unit after the index infarction and the second before hospital discharge, were obtained in 89% of the patients. In the remaining 11% of patients, one ECG was used. The designation of an infarction as "Q wave" or "non-Q wave" was determined by the development of diagnostic Q waves during the hospital period. All ECGs were coded for both diagnostic Q waves and ST segment elevation or depression, or both, according to location: anterior (leads V_2, V_3, V_4), lateral (leads I, aVL, V_5 or V_6) and inferior (leads III and aVF). Posterior infarction required an R/S ratio ≥1 in lead V_2. ST segment depression in lead V_2 associated with an upright T wave was coded as posterior. ST segment depressions were defined as deflections ≥1 mm from the baseline. Any coving of the ST segment consistent with a current of injury was considered ST segment elevation.

A radionuclide ejection fraction was obtained in 87% of patients during the initial hospital period. Chest X-ray films were obtained in the coronary care unit and read by the staff radiologists at each participating center. Pulmonary congestion was considered to be "present" (reflecting mild, moderate or severe congestion) or "absent." Similarly, the presence of pulmonary rales as recorded on the hospital chart any time in the coronary care unit was used for the variable "rales."

Patients were followed up at periodic intervals throughout the trial according to protocol. The minimal follow-up interval was 1 year, and the average duration of follow-up was 25 months.

End points. The trial's primary end points, mortality and first recurrent cardiac event, were verified by a four-member committee that reviewed all available information. The Hinkle-Thaler classification (14–16) was used to determine mechanism of death from cardiac causes. Criteria for nonfatal recurrent infarctions were the same as for the qualifying infarction, and all recurrent infarctions were reviewed by a separate three-member committee. A cardiac event was defined as the first occurrence of nonfatal infarction or cardiac death.

Statistical methods. Univariate relations were tested using the Fisher exact test, and event rates were evaluated using Kaplan-Meier curves (17) according to SAS LIFETEST (18), using the Wilcoxon statistic to compare the two curves. The Cox proportional hazards regression model (19) was calculated according to SAS PHREG (20). The data base utilized was version 3.0, released February 1990.

Results

More than 98% of the 1,234 study patients were followed up for at least 1 year. Six hundred seven patients (49%) demonstrated ST segment depression in one or more of the four ECG areas: 151 patients (25% of those with ST depression) had ST depression in the anterior leads with flat T waves or T wave inversion; 436 (72%) had ST depression in the lateral leads; 161 (27%) had ST depression in the inferior leads, and 120 (20%) had ST depression in the precordial leads associated with upright T waves representing posterior ischemia. Three hundred one patients (50%) had ST depression in more than one area.

Of the 953 patients who had ST segment elevation in one or more leads, 86% had diagnostic Q waves. Of the remaining 281 patients without ST elevation, those with ST depression did not develop diagnostic Q waves 71% of the time. The patients with neither ST elevation nor ST depression had a 76% incidence rate of non-Q wave infarction.

Overall, compared with patients without ST segment depression, patients with ST depression in any of the leads experienced more cardiac events and had a higher total mortality rate over the 52-month follow-up period (Fig. 1 A and B). The odds ratio was 1.6 ($p = 0.004$) for cardiac events in the year after the infarction and 1.9 ($p = 0.002$) for total mortality for patients with ST segment depression. Evaluation of postinfarction prognosis based on the location of ST depression showed that ST depression in the anterior, lateral or inferior locations was associated with increased mortality. The posterior location (ST depression in lead V_2 associated with upright T waves) demonstrated an event or mortality rate similar to that in patients with no ST depression (Table 1).

The prognosis for ST segment depression in patients with or without associated ST elevation in a separate ECG location is presented in Figure 2. Survival related to ST depression in patients with anterior or inferior ST elevation is presented in Figure 3. ST depression was a significant risk factor for late mortality whether or not ST elevation was present. At 1 year there was a 5.2% mortality rate in the 488 patients with ST elevation and no ST segment depression, compared with a 10.6% mortality rate in the 465 patients with ST elevation and ST depression in another region ($p = 0.0001$).

The effect was delayed but still significant in the absence of ST segment elevation. The 1-year mortality rate was 7.3% in patients without ST elevation or depression and 9.3% in patients with only ST depression. The difference increased

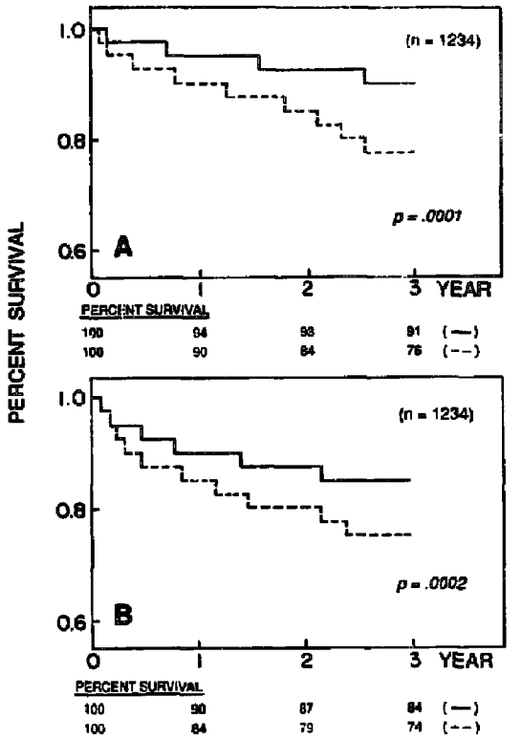

Figure 1. Probability of survival after myocardial infarction (**A**) and of survival free of cardiac events (**B**) in patients with (**dashed line**) or without (**solid line**) ST segment depression on the admitting electrocardiogram.

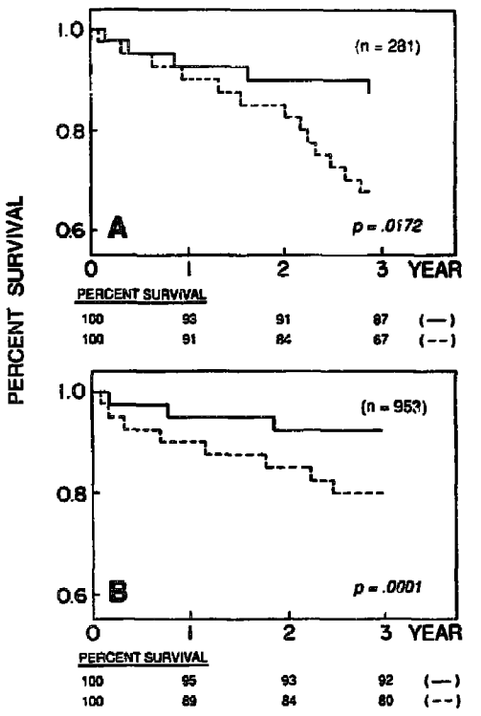

Figure 2. Cumulative survival in the clinical subgroups without (**A**) and with (**B**) ST segment elevation in any lead area, with (**dashed line**) or without (**solid line**) ST segment depression on the admitting electrocardiogram.

with longer follow-up (Fig. 1, p = 0.0172). Subgrouping patients according to the location of the ST elevation (Fig. 3) showed that the impact of ST segment depression was most striking in patients with ST elevation in inferior leads, where the mortality rate after 1 year was only 1.8% in patients with no ST depression compared with 11.0% if associated ST depression was present (p = 0.0001). A significant effect was also seen in patients with ST elevation in anterior leads who evidenced ST depression in the inferior or lateral leads, or both. The mortality rate was 6.9% in these patients if ST depression was not present compared with 13.6% if ST depression was present (p = 0.0005).

Univariate relations: ST segment depression and risk factors. The relations of ST segment depression to other important covariates for postinfarction mortality (7,12,13, 21,22) is presented in Table 2. There was a statistically significantly higher percent of women patients ≥70 years of age, patients with cardiac symptoms (New York Heart Association functional class >I) 1 month before the infarction, patients with rales during the index hospital period, patients with radiographic pulmonary congestion, patients with an average of >10 ventricular ectopic beats on a predischarge Holter recording, patients with left ventricular hypertrophy on the ECG and patients taking digitalis therapy in the group demonstrating ST segment depression.

Table 1. Site of ST Segment Depression in Relation to Death or Cardiac Events in 1 Year

	Patients (no.)	Mortality Rate (%)	Cardiac Event Rate (%)
No ST segment depression	627	5.6	10.3
ST depression site (ECG lead)			
Anterior (V_2, V_3, V_4)	151	11.5	17.6
Lateral (V_5, V_6, I, aVL)	436	10.4	16.5
Inferior (II, III, aVF)	161	11.2	16.8
Posterior (V_2, V_3)	120	6.7	8.4

When mortality was calculated according to the presence or absence of these clinical features associated with poor prognosis, however, patients with ST segment depression showed a higher mortality rate both in the presence or absence of each of the features tested (Table 3). The odds ratios for 1-year mortality rate and confidence intervals are presented in Figure 4.

ST segment depression was associated with increased mortality rate for patients with either non-Q wave or Q-wave infarctions, increasing from 7.2% for patients with non-Q wave infarctions without ST depression to 9.9% in patients with non-Q wave infarctions with ST depression (p = 0.026). In patients with Q wave infarctions, the mortality rate increased from 5.0% without ST depression to 10.5% when ST depression was present (p = 0.0001).

Multivariate analysis. The independent contribution of ST segment depression to determining total mortality or

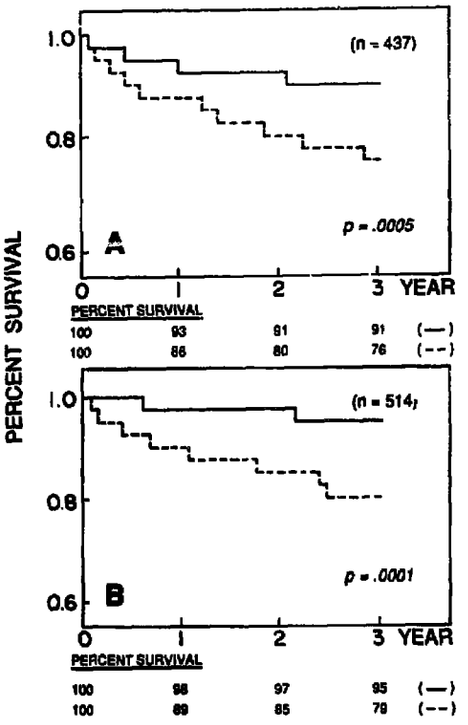

Figure 3. Cumulative survival of patients with ST segment elevation in anterior leads (A) and/or inferior leads (B), with (dashed line) or without (solid line) ST segment depression in other leads.

cardiac event rate was evaluated using Cox proportional hazard models (20). Each of the variables listed in Table 2 was evaluated (18) to determine the univariate relation to the end point total mortality and cardiac event (nonfatal infarction or cardiac death) rates. Previous infarction, pulmonary congestion, rales, age >70 years, functional class >I, ejection fraction <0.35%, history of hypertension, use of digitalis, peak creatine kinase <1,000 IU/dl, use of antiarrhythmic agents and ST segment elevation were all associated with an increased cardiac event rate ($p < 0.05$) and were all entered into the proportional hazards model for the cardiac event rate. These variables plus left ventricular hypertrophy were individually related to total mortality and were used to construct a model for total mortality. Adding the variable for ST segment depression in any location significantly improved the model for cardiac events (chi-square increased from 31.71 to 43.33; $p = 0.0007$) and for total mortality (chi-square increased from 29.28 to 39.39; $p = 0.0015$). In both models, for cardiac events and for total mortality, ST depression showed the highest individual independent relation to the respective end points ($p = 0.0007$ and 0.0015).

Table 2. Univariate Relations Related to ST Segment Depression

Clinical Feature	No ST↓ (%) (n = 627)	ST↓ (%) (n = 607)	p Value
Female	16.1	25.7	0.000
History of hypertension	35.9	40.6	0.090
Age ≥70 yr	9.7	15.2	0.004
Previous infarction	26.0	29.3	0.203
NYHA class >I	14.5	22.4	0.002
Q wave infarction	70.8	74.5	0.160
Rales above bases	37.3	43.8	0.023
Pulmonary congestion	16.1	25.1	0.000
>10 ventricular ectopic beats/h*	13.0	21.0	0.029
LVH on ECG	0.6	3.8	0.000
Digitalis treatment	11.8	16.3	0.027
Ejection fraction <0.35	18.8	19.2	0.877
Creatine kinase <1,000 IU/dl	53.1	49.3	0.185

*Holter monitor recordings were obtained in only 829 patients. ECG = electrocardiogram; LVH = left ventricular hypertrophy; NYHA class = New York Heart Association functional class; ST↓ = ST segment depression.

Table 3. Relation Between ST Segment Depression and Mortality

Clinical Variables	No ST↓ No.*	No ST↓ (%)†	ST↓ No.*	ST↓ %†	p Value‡
Anterolateral infarction	249	6.8	138	13.8	0.0009
Inferoposterior infarction	176	2.9	307	8.9	0.0004
First infarction	463	3.9	429	7.8	0.0001
Previous infarction	163	10.6	178	16.4	0.0057
NYHA class I	536	4.9	477	8.5	0.0001
NYHA class >I	91	10.0	130	17.0	0.0392
Q wave infarction	444	5.0	452	10.5	0.0001
Non-Q wave infarction	183	7.2	155	9.9	0.0262
Female gender	101	5.0	156	10.4	0.0085
Male gender	526	5.8	451	10.3	0.0001
No hypertension	402	4.5	360	7.9	0.0009
Hypertension	225	7.6	246	13.9	0.0001
Age <70 yr	566	5.2	515	8.6	0.0001
Age ≥70 yr	61	9.9	92	19.6	0.0320
No LVH	618	5.5	583	10.2	0.0001
No digitalis	553	4.0	508	8.2	0.0001
Digitalis	74	18.0	99	21.3	0.0427
No PVCs	373	3.2	316	6.7	0.0002
≥10 PVCs/h	56	9.1	84	16.8	0.1197
No pulmonary congestion	391	4.9	444	7.9	0.0004
Pulmonary congestion	99	9.2	149	17.7	0.0028
No rales above bases	391	4.9	340	5.9	0.0719
Rales above bases	233	6.9	265	16.0	0.0001
Ejection fraction ≥0.35	441	3.9	437	5.8	0.0012
Ejection fraction <0.35	102	15.9	104	27.9	0.0021
Creatine kinase <1,000 IU/dl	326	5.4	290	14.2	0.0001
Creatine kinase ≥1,000 IU/dl	294	5.8	307	6.6	0.0459

*Number of patients with indicated variable. †1-year mortality rate calculated from life-table curves. ‡Calculated from Kaplan-Meier curve, Wilcoxon statistic. LVH = left ventricular hypertrophy (voltage and ST-T criteria); NYHA class = New York Heart Association functional class; PVCs = premature ventricular complexes.

Discussion

These results emphasize the increased risk for late mortality and cardiac events in patients after a myocardial infarction associated with ST segment depression on the coronary care unit ECG. ST segment depression in any lead group, with or without ST elevation elsewhere on the ECG, was associated with increased total mortality and an increase

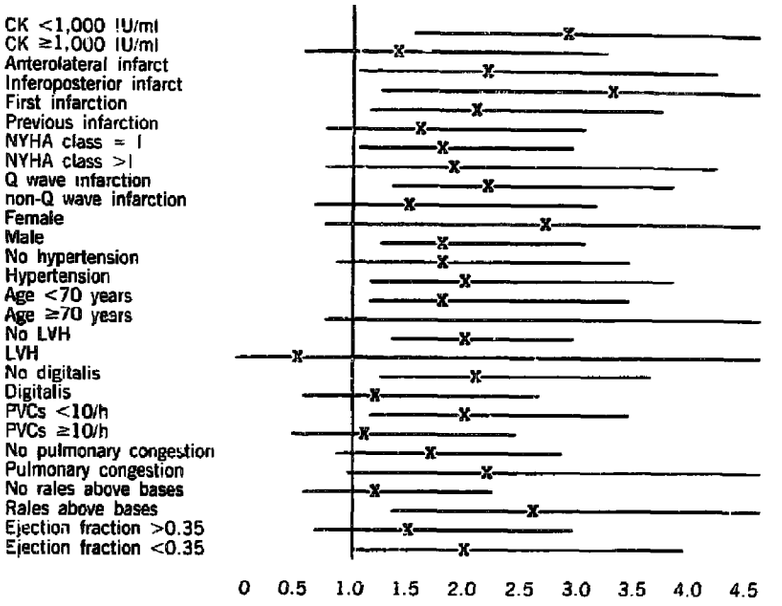

Figure 4. Odds ratios of 1-year mortality risk with and without ST segment depression for indicated variables. Width of bar represents 95% confidence intervals. CK = creatine kinase; LVH = left ventricular hypertrophy; NYHA class = New York Heart Association functional class; PVCs = premature ventricular complexes.

in new cardiac events over the 52-month follow-up period. Patients with ST segment depression were more likely to be women, be aged ≥70 years, have ECG left ventricular hypertrophy, have cardiac symptoms in the month preceding the acute infarction, take digitalis, have clinical rales, have >10 ventricular ectopic beats/h and have congestion on the chest X-ray film obtained in the coronary care unit (Table 2).

All of these variables have been associated with increased mortality after a myocardial infarction (21); however, ST segment depression was an independent predictor for increased mortality and cardiac events. When these variables were evaluated using a Cox proportional hazards model (20), ST segment depression was independently associated with all causes of mortality or new cardiac events, or both, after controlling for the other risk factors.

Electrocardiographic subgroups with ST depression. Several investigators have looked at specific subgroups of patients with ST segment depression. Willems et al. (23) showed that ST segment depression >2 mm in any of the inferior leads in the context of anterior ECG ST segment elevation was associated with an increase in mortality rate from 6.7% in patients with anterior infarction without ST segment depression to 14.3% in patients with such ST segment changes. These investigators also showed a similar effect in patients with inferior infarction with anterior ST segment depression (23). Our data support these findings: We observed that patients with ST segment elevation in the inferior leads had a much worse prognosis if they had ST segment depression in the anterior or lateral leads (11.3% 1-year mortality rate) than if no ST segment depression was present (1.8% 1-year mortality rate, $p = 0.0001$; Table 2).

Other investigators have shown the importance of ST segment depression as a predictor of increased mortality in selected myocardial infarction subsets (5,6,24–27). Schechtman et al. (3) noted in patients with non-Q wave infarction a marked increase in 1-year mortality rate in those with ST segment depression; 1-year mortality rate increased from 5.5% in patients with no ST depression on serial ECGs to 10.1% in those with ST depression on either admission or discharge and increased to 22.2% in patients with ST depression on both ECGs. In another study, Schechtman et al. (28) found that ST segment depression was associated with mortality at 3 months but not with later mortality. We showed the importance of ST segment depression in major ECG subgroups of acute infarction: anterior and inferior and Q wave and non-Q wave subgroups. In our study, the difference in survival between patients with and without ST depression appeared to increase after the 1st year.

Willich et al. (29) and Hollander et al. (4) also showed a poor outcome in patients with acute infarction presenting with ST segment depression. Crenshaw et al. (30) showed that ST segment depression was associated with a higher prevalence of clinical risk factors (diabetes mellitus, hypertension, previous myocardial infarction and older age) and a higher mortality rate in patients who underwent coronary angiography.

Coronary artery studies. Patients with left circumflex coronary artery or branch occlusion commonly present with ST segment depression (31–35). Other studies have suggested that ST segment depression is frequently associated with extensive coronary artery disease. Sclarovsky et al. (36), in a small study of patients with unstable angina, showed that 7 of 10 patients with ST depression and T wave inversion had left main coronary artery disease. Raunio et al. (37), in an autopsy study, found that 89% of patients who died with ST segment depression as the primary ECG abnormality had severe three-vessel disease.

Anterior precordial ST segment depression. Anterior precordial ST segment depression in the presence of inferior ST elevation has been extensively studied. Ruddy et al. (38),

using gated blood pool scans and thallium-201 perfusion imaging, concluded that patients with this combination had more severe wall motion impairment and greater hypoperfusion of the inferior and adjacent segments than did patients with inferior ST changes alone. This result confirmed and extended the earlier observations of Shah et al. (5) and Lew et al. (39). Roubin et al. (40), in contrast to Lew et al. (39), noted that patients with inferior infarction and associated anterior ST segment depression had a higher incidence of left anterior descending artery stenosis (36%) than that of patients with inferior infarction and no ST segment depression (6%); their data, like that of Lew et al. (39) also indicated that patients with single-vessel disease had larger infarct-related arteries supplying the lateral wall as well as the inferior wall of the left ventricle. Gibson et al. (41) and Berland et al. (42) also concluded that the amount of ischemia and infarction was greater in the patients with precordial ST depression. The question of whether anterior ST segment depression is associated with a worse prognosis in patients with their first inferior myocardial infarction is being analyzed separately using the entire MDPIT data base (F. I. Marcus, personal communication.)

The difficulty of distinguishing between anterior subendocardial ischemia and posterior transmural injury in patients with anterior ST segment depression was summarized by Boden and Spodick (43). The key role of T wave inversion in locating the ischemia on the anterior wall or on the posterior wall if the T wave remained upright was emphasized. Our data emphasize the importance of this distinction. "Posterior ST segment changes" in our patients were associated with a 1-year mortality rate <7%, whereas "anterior" changes were associated with a higher mortality rate (Table 1). If ST segment depression was associated with ST segment elevation in the inferior leads, the 1-year mortality rate increased an astounding six times from 1.8% to 11.0% ($p = 0.0001$).

Anterior infarction. A similar worsening of prognosis of patients with anterior infarction was observed in the presence of ST segment depression. If ST depression was noted in the inferior or lateral leads, or both, in patients with anterior injury reflected on the ECG, the 1-year mortality rate was 13.8% compared with 7.0% if no additional ST depression was present ($p = 0.022$; Fig. 3).

Implications for thrombolytic therapy. Several studies have suggested that thrombolytic therapy is less effective in patients with only ST segment depression than in patients with ST elevation. In the Second International Study of Infarct Survival study (10), 212 patients with only ST depression were randomized to placebo or streptokinase therapy. In those patients treated with streptokinase, the mortality rate was 18.7% compared with 18.6% for those randomized to placebo. In view of the work of Macdonald et al. (44) and Yasue et al. (45), this outcome might be predicted. These investigators showed in separate models that occlusion of an artery resulting in ST segment depression was usually associated with more severe underlying stenosis and the frequent development of collateral vessels. Conversely, occlusion of an artery without preexisting severe obstruction would lead to a pattern of ST elevation. Thrombolytic therapy would be expected to be most effective if lysis of the clot restored flow through a minimally stenotic artery. The work cited suggests that ischemia presenting with ST segment depression would be the result of more severe coronary stenosis.

Conclusions. The increased mortality associated with ST segment depression was observed in patients with both Q wave and non-Q wave infarction and was seen in both anterior and inferior locations. From the demonstration that ST depression is correlated with multiple clinical features associated with increased risk, as well as the available anatomic and physiologic observations, we hypothesize that ST segment depression associated with acute infarction is a manifestation of more extensive coronary artery disease or more widespread ischemia, or both. This could be the result of multivessel disease or larger occluded infarct-related arteries. Further characterization of patients with ST depression, especially the coronary anatomy, would be useful to determine whether acute interventional strategies can be utilized to improve prognosis.

We thank Arthur J. Moss, MD, FACC for his leadership in the performance of this study and helpful comments on this report and Frank I. Marcus, MD, FACC for his careful review of the manuscript.

References

1. Krone RJ. The role of risk stratification in the early management of a myocardial infarction. Ann Intern Med 1992;116:223–37.
2. Capone RJ, Pawitan Y, El-Sherif N, et al. Events in the cardiac arrhythmia suppression trial: baseline predictors of mortality in placebo-treated patients. J Am Coll Cardiol 1991;18:1434–8.
3. Schechtman KB, Capone RJ, Kleiger RE, et al. Risk stratification of patients with non-Q wave myocardial infarction: the critical role of ST segment depression. Circulation 1989;80:1148–58.
4. Hollander G, Ozick H, Greengart A, Shani J, Lichstein E. High mortality early reinfarction with first nontransmural myocardial infarction. Am Heart J 1984;108:1412–6.
5. Shah PK, Pichler M, Berman DS, et al. Noninvasive identification of a high risk subset of patients with acute inferior myocardial infarction. Am J Cardiol 1980;46:915–21.
6. Hlatky MA, Califf RM, Lee KL, Pryor DB, Wagner GS, Rosati RA. Prognostic significance of precordial ST-segment depression during inferior acute myocardial infarction. Am J Cardiol 1985;55:325–9.
7. Tibbits PA, Evaul JE, Goldstein RE, et al. Serial acquisition of data predict one-year mortality rate after acute myocardial infarction. Am J Cardiol 1987;60:451–5.
8. Schweitzer P. The electrocardiographic diagnosis of acute myocardial infarction in the thrombolytic era. Am Heart J 1990;119:642–54.
9. Gruppo Italiano per lo Studio della Streptochinasi nell'Infarto Miocardico (GISSI). Effectiveness of intravenous thrombolytic treatment in acute myocardial infarction. Lancet 1986;1:397–401.
10. ISIS-2 (Second International Study of Infarct Survival) Collaborative Group. Randomized trial of intravenous streptokinase, oral aspirin, both or neither among 17,187 cases of suspected acute myocardial infarction: ISIS-2. Lancet 1988;2:349–60.
11. ISIS-3 (Third International Study of Infarct Survival) Collaborative Group. ISIS-3: a randomized comparison of streptokinase vs tissue plasminogen activator vs anistreplase and of aspirin plus heparin vs

aspirin alone among 41,299 cases of suspected acute myocardial infarction. Lancet 1992;339:753–70.

12. Multicenter Diltiazem Postinfarction Trial Research Group. The effect of diltiazem on mortality and reinfarction after myocardial infarction. N Engl J Med 1988;319:385–92.
13. Greenberg H, Gillespie J, Dwyer EM Jr, and the Multicenter Post-Infarction Research Group. A new electrocardiographic classification for post-infarction clinical trials. Am J Cardiol 1987;59:1057–63.
14. Hinkle LE, Thaler HT. Clinical classification of cardiac deaths. Circulation 1982;65:457–64.
15. Marcus FI, Friday K, McKans J, et al. Age-related prognosis after myocardial infarction (The Multicenter Diltiazem Postinfarction Trial). Am J Cardiol 1990;65:559–66.
16. Marcus FI, Cobb LA, Edwards JE, et al. Mechanism of death and prevalence of myocardial ischemic symptoms in the terminal event after myocardial infarction. Am J Cardiol 1988;61:8–15.
17. Kaplan EL, Meier P. Nonparametric estimation from incomplete observations. J Am Stat Assoc 1958;53:457–81.
18. SAS Institute, Inc. Additional SAS/STAT Procedures, release 6.03. Cary, NC: SAS Institute, Inc., 1988:49–90; SAS Technical Report P-179.
19. Cox DR. Regression models and life-tables. J R Stat Soc [B] 1972;34:187–220.
20. SAS Institute, Inc. SAS/STAT Software: The PHREG Procedure, version 6. Cary, NC: SAS Institute, Inc., 1991:1–59.
21. The Multicenter Postinfarction Research Group. Risk stratification and survival after myocardial infarction. N Engl J Med 1983;309:331–6.
22. Piérard LA, Dubois C, Albert A, Chapelle J-P, Carlier J, Kulbertus H. Prognostic significance of a low peak serum creatinine kinase level in acute myocardial infarction. Am J Cardiol 1989;63:792–6.
23. Willems JL, Willems RJ, Willems GM, et al. Significance of initial ST segment elevation and depression for the management of thrombolytic therapy in acute myocardial infarction. Circulation 1990;82:1147–58.
24. Lembo NJ, Starling MR, Dell'italia LJ, Crawford MH, Chaudhuri TK, O'Rourke RA. Clinical and prognostic importance of persistent precordial (V_1–V_4) electrocardiographic ST segment depression in patients with inferior transmural myocardial infarction. Circulation 1986;74:56–63.
25. Gelman JS, Saltups A. Precordial ST segment depression in patients with inferior acute myocardial infarction: clinical implications. Br Heart J 1982;48:560–5.
26. Herlitz J, Hjalmarson A. Occurrence of anterior ST depression in inferior myocardial infarction and relation to clinical outcome. Clin Cardiol 1987;10:529–34.
27. Bates ER, Clemmensen PM, Califf RM, et al. Precordial ST segment depression predicts a worse prognosis in inferior infarction despite reperfusion therapy. J Am Coll Cardiol 1990;16:1538–44.
28. Schechtman KB, Capone RJ, Kleiger RE, et al. Differential risk patterns associated with 3 month as compared with 3 to 12 month mortality and reinfarction after non-Q wave myocardial infarction. J Am Coll Cardiol 1990;15:940–7.
29. Willich SN, Stone PH, Muller JE, et al. High-risk subgroups of patients with non-Q wave myocardial infarction based on direction and severity of ST segment deviation. Am Heart J 1987;114:1110–9.
30. Crenshaw JH, Mirvis DM, El-Zeky F, et al. Interactive effects of ST-T wave abnormalities on survival of patients with coronary artery disease. J Am Coll Cardiol 1991;18:413–20.
31. Pruitt RD, Dennis EW, Kinard SA. The difficult electrocardiographic diagnosis of myocardial infarction. Prog Cardiovasc Dis 1963;6:8–106.
32. Blanke H, Cohen M, Schlueter GU, Karsch KR, Rentrop KP. Electrocardiographic and coronary arteriographic correlations during acute myocardial infarction. Am J Cardiol 1984;54:249–55.
33. Sclarovsky S, Topaz O, Rechavia E, Strasberg B, Agmon J. Ischemic ST segment depression in leads V_2–V_3 as the presenting electrocardiographic feature of posterolateral wall myocardial infarction. Am Heart J 1987;113:1085–90.
34. Boden WE, Kleiger RE, Gibson RS, et al. Electrocardiographic evolution of posterior myocardial infarction: importance of early precordial ST-segment depression. Am J Cardiol 1987;59:782–7.
35. Mamby SA, Bradley AB, Boden WE. Early precordial ST-segment depression due to isolated acute right or left circumflex coronary artery occlusion. Am J Cardiol 1987;60:726–8.
36. Sclarovsky S, Rechavia E, Strasberg B, et al. Unstable angina: ST segment depression with positive versus negative T wave deflections—clinical course, ECG evolution, and angiographic correlation. Am Heart J 1988;116:933–41.
37. Raunio H, Rissanen V, Helin M, Rehnberg S, Romppanen T, Janatuinen E. Early pronounced ST segment depression with marked J point decline heralding acute lethal clinical myocardial infarction: necropsy electrocardiographic correlative study. Am Heart J 1982;103:32–7.
38. Ruddy TD, Yasuda T, Gold HK, et al. Anterior ST segment depression in acute inferior myocardial infarction as a marker of greater inferior, apical, and posterolateral damage. Am Heart J 1986;112:1210–6.
39. Lew AS, Weiss AT, Shah PK, et al. Precordial ST segment depression during acute inferior myocardial infarction: early thallium-201 scintigraphic evidence of adjacent posterolateral or inferoseptal involvement. J Am Coll Cardiol 1985;5:203–9.
40. Roubin GS, Shen WF, Nicholson M, Dunn RF, Kelly DT, Harris PJ. Anterolateral ST segment depression in acute inferior myocardial infarction: angiographic and clinical implications. Am Heart J 1984;107:1177–82.
41. Gibson RS, Crampton RS, Watson DD, et al. Precordial ST-segment depression during acute inferior myocardial infarction: clinical, scintigraphic and angiographic correlations. Circulation 1982;66:732–41.
42. Berland J, Cribier A, Behar P, Letac B. Anterior ST depression in inferior myocardial infarction: correlation with results of intracoronary thrombolysis. Am Heart J 1986;111:481–8.
43. Boden WE, Spodick DH. Diagnostic significance of precordial ST-segment depression. Am J Cardiol 1989;63:358–61.
44. Macdonald RG, Hill JA, Feldman RL. ST segment response to acute coronary occlusion: coronary hemodynamic and angiographic determinants of direction of ST segment shift. Circulation 1986;74:973–9.
45. Yasue H, Omote S, Takizawa A, et al. Comparison of coronary arteriographic findings during angina pectoris associated with S-T elevation or depression. Am J Cardiol 1981;47:539–46.